UNITED STATES DISTRICT COURT
DISTRICT OF MINNESOTA

IN RE NATIONAL HOCKEY LEAGUE PLAYERS' CONCUSSION INJURY LITIGATION	)	MDL No. 14-2551

AFFIDAVIT OF ANN C. MCKEE, M.D.

Ann C. McKee, M.D., being duly sworn, deposes and states that:

1. I am a Professor of Neurology and Pathology at Boston University School of Medicine (BUSM), Associate Director of the Boston University Alzheimer's Disease Center (BU ADC) and Director of the Neuropathology Core of the BU ADC. In addition, I am the Director of the CTE Center for the BU ADC and the Director of the CTE Center's Brain Bank (VA-BU-CLF brain bank), a collaborative project involving the United States Department of Veteran's Affairs (VA), the University, and the Concussion Legacy Foundation (CLF), formerly the Sports Legacy Institute (SLI).

2. I am a board-certified neurologist and neuropathologist and the principal investigator on several ongoing and completed research projects investigating the progressive neurodegenerative disease, Chronic Traumatic Encephalopathy (CTE). My curriculum vitae is appended as Exhibit A.

3. Boston University's Alzheimer's Disease Center was established in 1996 as one of 29 centers funded by the National Institutes of Health to advance research on Alzheimer's disease and related conditions. The BU ADC, through its CTE Center, also supports high-impact, innovative research on CTE and other long-term consequences of repetitive brain trauma in athletes and military personnel.

4. The CTE Center was founded in 2008 under a collaboration with the non-profit Sports Legacy Institute (currently, Concussion Legacy Foundation). This formal collaboration ended in 2014, and the CTE Center is now an independent Boston University academic research center whose mission is to conduct state-of-the-art research on CTE, including its neuropathology and pathogenesis, clinical presentation, genetics and other risk factors, biomarkers, methods of detection during life, and methods of prevention and treatment.

5. As part of the CTE Center, the Brain Bank was created in 2008 at the Edith Nourse Rogers Memorial Veterans Hospital in Bedford, Massachusetts ("VA Hospital") in collaboration with the VA. The purpose of the VA-BU-CLF Brain Bank is to collect and study post-mortem human brain and spinal cord tissue to better understand the effects of trauma on the human nervous system. Donated tissue is stored in the VA-BU-CLF Brain Bank for use in studies conducted at the CTE Center as well as for studies conducted by or in collaboration with other research laboratories around the world.

6. Research discoveries made by the CTE Center are published in a variety of peer-reviewed publications and have been widely cited by scientific leaders throughout the world. Many organizations, including the National Football League and the National Football League Players Association, have voiced support for CTE Center research and have encouraged athletes to participate when possible.

Background on Chronic Traumatic Encephalopathy Research

7. Chronic Traumatic Encephalopathy (CTE) was first reported in 1928 by Harrison Martland, a New Jersey pathologist. Martland described the clinical aspects of a progressive neurological deterioration that occurred after repetitive brain trauma in boxers. He referred to this condition as "punch drunk," but other terms were introduced over the decades that followed, including "traumatic progressive encephalopathy" and "dementia pugilistica." By the 1940s, the term "chronic traumatic encephalopathy" was used, recognizing that the condition could arise from brain trauma of a variety of sources in both men and women. CTE has been clinically associated with symptoms of irritability, impulsivity, aggression, depression, short term memory loss and heightened suicidality. These associated symptoms typically appear 8-10 years following reported accounts of repetitive mild traumatic brain injury.

8. Currently, we understand that CTE is a neurodegeneration characterized by the abnormal accumulation of hyperphosphorylated tau protein (p-tau) within the brain. Tau proteins are proteins that stabilize microtubules. They are abundant in neurons of the central nervous system. When tau becomes hyperphosphorylated, it forms neurofibrillary tangles (NFT), causing it to aggregate, or group, in an insoluble form. This insoluble accumulation interferes with normal neuronal function and can lead to cell death. In early stages of the disease, NFTs appear to be clustered in distinct locations of the brain and, as CTE becomes more advanced, widespread brain regions become affected. This allows progressive staging of pathology and correlation of pathology findings with reported clinical symptoms. Like many other neurodegenerative conditions, CTE, at present, can only be definitively diagnosed by post-mortem examination of brain tissue, although significant efforts are

underway to improve clinicians' ability to use available diagnostic tools to evaluate for the presence of early to late stage CTE during life.

9. In March 2013, the National Institutes of Health (NIH), supported by the Foundation for NIH's Sports Health Research Program with funding from the National Football League, launched an effort to define the neuropathological characteristics of CTE. One of the initial projects was to convene two consensus meetings of expert neuropathologists to define, as a group, the neuropathological criteria for the diagnosis of CTE, and to distinguish it from pathologies of other neurodegenerative diseases associated with tau protein aggregation (known as "tauopathies"), including Alzheimer's Disease. This panel of expert neuropathologists met in 2015 and 2016. Using digitized images of 11 cases of CTE from the BU CTE Center brain bank, they found that the p-tau pathology of CTE is unique and can be easily distinguished from other tauopathies.

10. According to the NIH consensus panel, the defining lesion of CTE or its *pathognomonic* lesion, consists of an accumulation of abnormal tau in neurons and astroglia distributed around small blood vessels at the depths of sulci in the cortex of the brain in an irregular pattern. Supportive features of CTE were also identified and defined.[1] The panel noted that, thus far, CTE has only been found in individuals who were exposed to brain trauma, typically multiple episodes. The consensus panel's determinations validated the preliminary diagnostic criteria reported by McKee et al (2013) and confirmed the criteria

[1] Supportive features include abnormal p-tau immunoreactive pretangles and NFTs preferentially affecting superficial layers, pretangles, NFTs or extracellular tangles primarily in CA2 and CA4 of the hippocampus, NFTs in subcortical nuclei, including the mammillary bodies and other hypothalamic nuclei, amygdala, nucleus accumbens, thalamus, midbrain tegmentum, isodendritic core, p-tau immunoreactive thorned astrocytes at the glial limitans in the subpial and periventricular regions, p-tau immunoreactive large grain-like and dot-like structures, and TDP-43 immunoreactive neuronal cytoplasmic inclusions and dot-like structures in the hippocampus, anteromedial temporal cortex and amygdala.

always used by the CTE Center for the diagnosis of CTE when evaluating its donor brain tissue.

11. A second NIH consensus panel met in 2016 and evaluated the digital slides of 29 cases of CTE from the BU CTE Center. The second panel confirmed the original panel's findings, and further characterized the staging of pathological severity.

12. In addition to the consensus panel's determinations, the CTE Center has been actively conducting research on the clinical presentation and symptoms of CTE, the risks associated with playing contact sports, the risks of beginning to play sports at a young age, genetic modifiers of the disease, co-morbidities, and its pathological progression. The BU CTE Center has been remarkably productive (more than 60 peer-reviewed manuscripts since 2008). Nonetheless, there are numerous questions still left to be answered. The uninterrupted progression of ongoing work by the CTE Center and the VA-BU-CLF Brain Bank is critical to finding these answers particularly because these answers impact public health for athletes as well as military veterans.

Research Participant Recruitment, Brain Donation and Confidentiality

13. As Director of the CTE Center and Director of the VA-BU-CLF Brain Bank, I manage the brain donation program. I am also currently the principal investigator on a U01 project funded by NINDS and the National Institute of Biomedical Imaging and Bioengineering (NIBIB), entitled "Understanding Neurologic Injury and Traumatic Encephalopathy" (UNITE). UNITE examines the neuropathology and clinical presentation of brain donors who, based on prior athletic or military exposure, are designated as at risk for the development of CTE.

14. I oversee the process of subject recruitment and brain donation. For a majority of brain donors, the subjects' next of kin or legally authorized representative (LAR) contact the Brain Bank and agree to donate. While living, some study subjects agree to donate their brain and spinal cord after death. But the next of kin or LAR is still asked to consent to the donation at death, and is assured that the donor's name will not be disclosed, and that no personally identifiable information will be used or disclosed. This statement is also included on the CTE Center website. In addition, consent forms executed by living participants in UNITE, and required by the University's Institutional Review Board (which evaluates human subject and other research pursuant to federal regulatory requirements), promise confidentiality and prohibit the sharing of a participant's identifiable information with third parties.

15. Next of kin and LARs frequently express their concern, and anxiety, about the confidentiality of the information that might be uncovered as part of the CTE Center's research. Family members often tell us that they do not want the deceased donor's identity being uncovered, either directly or through deduction. This is particularly true with regard to personal information disclosed as part of a clinical interview(s) with the donor prior to death or with his/her family after death. Family members and donors understand that such information, if disclosed, will allow for third party identification by deduction. This information includes, but is not limited to, an individual's status as a professional athlete, the number of years he/she played, the position he/she played as part of sports, major medical events, the experiences he/she had earlier in life and other biographical data specific to a particular donor. (Other families and donors allow us to publicly announce our research results, but we do not make such public statements without express authorization to do so.)

16. My colleagues in the CTE Center and I have taken great care to preserve participant and donor confidentiality. My research depends on the trust that the donors, and their families, place in us. It is an incredible privilege to be entrusted with another individual's brain, and to be given an accurate glimpse of their life and the personal moments, some of which were quite difficult, that were a part of it. Our promise of absolute privacy and confidentiality is critical to preserving this trust. If potential donors and their families fear that they will become embroiled in litigation or that their reputations, or those of their deceased loved ones, might be harmed if their identities become known as part of litigation, we could not successfully continue the VA-BU-CLF Brain Bank or the Brain Registry. As a result, much of my work, including all our work on CTE and finding answers to the critical questions surrounding athlete and veteran safety, would be threatened.

Brain Specimen Preparation and Deidentification

17. Gathering the primary research materials responsive to the NHL Subpoena, and conducting the necessary deidentification process, *for just the research I oversee alone,* poses not only an insurmountable burden, but also is impractical. Compliance becomes virtually impossible when we consider all of the CTE-related research conducted by faculty and scientists affiliated with the CTE Center. A third party could not reduce this burden or conduct any of the necessary steps on our behalf, because the consents we currently have in place strictly limit access of private, confidential information to Boston University researchers. While consents do allow for third party researchers to be provided with access to samples for the advancement of research, education, science or therapy, this consent is limited to the provision of samples on a deidentified basis only. In addition, the materials are housed at the VA Hospital under an agreement with Boston University. The VA Hospital

is a HIPPA-covered health care facility and third party access, even if restricted to the research space, raises significant additional confidentiality concerns unrelated to the Brain Bank itself.

18. While confidentiality is one important reason that third party offers to "share this burden" are untenable, the other is that the volume of information, the fragility of many of the items at issue and their importance to ongoing and future research cannot be overstated. Moreover, I take my role as steward of the research materials under my control seriously, along with my promises to preserve anonymity, and, accordingly, would have to participate and review every piece of information that left my laboratory to ensure that it complies with the protections we have put into place to preserve the trust of our donors.

19. To better understand what is being asked of the CTE Center, the VA-BU-CLF brain bank protocol is appended as Exhibit B. The brain bank contains approximately 400 brains from donors. The protocol calls for the production of approximately ~172,000 gross photographs of each brain and spinal cord. Each of the individual photographs contain, within the image, a marker designating the autopsy number of the donor. The autopsy number is considered a patient identifier, and under no circumstances are we allowed to give out the autopsy number of a subject. HIPAA regulations state that we must eliminate the autopsy number and replace it with a de-identified "pincode" that cannot be traced back to any specific patient before we give out any materials. To eliminate the autopsy number from each individual photograph would require approximately 10 minutes per photograph; assuming a "de-identification" rate of six photographs per hour, this process would take more than 28,600 hours, or, assuming a 40-hour work week, in excess of 13 years. In addition, the protocol produces approximately ~120,000 photomicrographs of stained slides of the brain and spinal cord of donors, which also contain identifiers. The preceding time line assumes a

third party has the technical expertise necessary to perform these tasks; if not, even more time will be needed to complete the de-identification process. The process of merely locating nearly 400,000 photographs and digital photographs, gathering them into secure boxes or copying them onto CDs to give to a third party would require an estimated additional 4 hours per brain donor.

20. Under the protocol, we also have 264,000 glass microscopy slides and 40,000 large landscape glass slides containing sections from the brain donors. I estimate that locating and packaging these glass slides for transport would require a minimum of 4000 hours. Because these glass slides hold human tissue, the transfer to a third party would also require authorization. Most donors have consented to the transfer of donated *tissue samples* to third parties only if the sample is de-identified. As a rule, brain banks do not give out their primary data, that is, the actual glass slides that were produced by the brain bank laboratory, as that would risk losing their primary data forever. Primary data must remain under the jurisdiction of the original research lab. Transfer of other *tissue samples* to other laboratories is done if it is for the advancement of research, education, science or therapy and if the researcher requesting the samples is a NIH-funded investigator with approved research credentials. Moreover, as the attached protocol shows, the preparation of the glass slides represents significant time and resources. The slides are fragile and carry significant risk of breakage with packaging and transport. If lost or damaged, the slides cannot be replaced and critical source material from individual donors could be lost forever.

21. The process of de-identifying and removing the primary research materials from the VA facility would be highly disruptive to both my laboratory and to the VA facility itself. In practical terms it would shut down my research. It would require untold people-power, which I do not have access to, and around the clock effort, likely made by scientists

and research colleagues whose time would be much better spent towards advancing important research questions and discovery. And the concerns I have described do not even factor in the time it would take to de-identify the clinical reports conducted by and under the direction of my colleague Dr. Robert Stern, who discusses that topic in detail in his affidavit.

22. Most critically, the removal of the research materials requested by the NHL Subpoena prevents their usage in ongoing and future research endeavors. This includes not only my research, but also the research of my colleagues and my collaborators around the world that rely on the work we do as part of larger collaborative studies. The ripple effect of inaccessibility of such data, even if time limited, is unfathomable.

23. Following all this effort toward de-identification, we would ultimately be left with slides, photographs and largely redacted reports that contain little to no information linking the neuropathological findings to biographical information of an individual. For example, I would expect one could not even discern from redacted reports whether data are associated with professional hockey players, professional football players or with an individual that never played a sport in his/her life given the high profile nature of any donor's professional status. Scientifically, I have difficulty understanding what new information either party to the NHL Players' Litigation would glean from such materials.

Heightened Importance of Preserving Integrity of Sensitive Research

24. Scientific debate and discussion are critical to research advancement. Hypotheses get challenged, methodologies critiqued and, through this, outputs get improved and built on. The aim of all of our CTE-related research is to publish our findings. Publication is an essential prerequisite to professional advancement, future funding opportunities and the reputational success of any scientist. More importantly, publication

allows for sharing information with the public at large and with peers all over the world who can use it to advance their own research program or, as the case may be, to challenge its underlying interpretations. Methodologies are shared to allow third-party replication. The CTE Center occasionally approves the sharing of brain tissue with other researchers, on a deidentified basis, conditioned on its utilization to further scientific research.

25. Prior to submission of an article reporting on original research, however, scientists often crowdsource their ideas and analyses in private correspondence—through laboratory meetings, hallway discussions, the exchange of results and drafts and, at times, through active debate. The NHL Subpoena's invasive demand for all CTE-related pre-publication discussions, including with peer reviewers retained by journals, threatens the foundation on which science thrives. BU's lawyers have told me that the NHL, in its legal brief, has narrowed that request only to "published publications," but that does not really minimize the scope of this invasive request. If individuals worry that any scientific discussion, question or edit made to a *draft* article could be picked apart at a later date by a litigant to serve its own needs, open and frank discussion becomes vulnerable. Science cannot thrive under the cloud of such uncertainty. It will diminish the quality, pace and breadth of our work and will negatively impact the entire field of study.

26. For CTE-related research, as for any scientific question that directly impacts large, multi-billion dollar industries, the stakes of preventing this "chilling" effect are even higher. It could discourage new talent from entering the field or experts from asking questions and providing unvarnished scientific answers. The science would inevitably slow, due to the disruptive effect of the litigation and the reduction in those willing to risk being dragged into it. This could discourage the influx of new talent, new sponsors and new donors. This would be detrimental to society at large, as additional knowledge of the science

of CTE can only benefit the individuals, institutions and industries impacted by its devastating effects.

27. All major scientific journals subject their articles to a robust, largely effective, review by scientific experts in the field in question prior to publication. This process is confidential to ensure that scientific reviewers can speak freely with a unifying goal of improving the scientific output. Similar to my preceding comments regarding the chilling effects of producing private scientific communications, production of peer reviewer comments on pre-publication manuscripts could also undercut the integrity of the research enterprise. Without question, this will negatively impact the quality of the work produced, the willingness of scientists (who volunteer for this role) to serve as peer reviewers and the trust the public has in an enterprise that we all rely on to advance medical knowledge.

Response to NHL Position

28. I have read Dr. Rudy Castellani's affidavit, submitted with the NHL's Memorandum of Law in Support of its Motion to Compel Production**.** In paragraph 12, Dr. Castellani states that "most publications depict microscopic pathology deemed "representative . . . in support of the case and the hypothesis," and that he would like access to copies of gross pathology photographs, all brain slides, and clinical data so he can "verify the accuracy of the reports, evaluate for other pathological processes that may be significant, and conduct a full, independent neuropathological analysis of the cases." This is not the way science works or should work. First, as I hope the foregoing paragraphs have demonstrated, I am required to submit representative images by the journals given the enormous volume of data we generate in support of our conclusions. Almost all manuscripts, in any field, do so. Second, if I provide the thousands of slides and images I maintain to every researcher who

doesn't understand or, for whatever reason, doesn't believe in my scientific conclusions or has a belief that he/she could 'do the science better', there would be no ability for my lab to actually conduct studies that progress science. Instead, my papers go through a rigorous scientific review by *scientific experts in the field* prior to their publication. I respond to any questions they may have and, as necessary, provide supplemental data and materials to verify to these experts every conclusion reached. It is apparent that Dr. Castellani, who is well-known in the field for his belief that there is no link between concussions and CTE, wants to undermine my *peer-reviewed, accepted research conclusions*.

29. Notwithstanding my frustration with the implications, I would not object to providing Dr. Castellani with the material he describes in paragraph 13 of his affidavit with respect to the late Lawrence Zeidel. My research colleague examined Mr. Zeidel's brain and made a pathological diagnosis of CTE. I am confident that any neuropathologist who reviews the data with a neutral view will reach the same conclusion. I understand and acknowledge that Mr. Zeidel's Estate is a participant in this lawsuit, and for that reason, his case takes on added significance. His Estate has authorized the CTE Center to disclose certain information which has already been provided to the NHL's lawyers. However, for the reasons I have described in this affidavit, I strenuously object to providing the NHL, Dr. Castellani, or any researcher, information on any other research subject whose family has not consented to the intrusive disclosure sought by the NHL. And, for the reasons I have described in this affidavit, I urge the Court to consider the devastating impact that the open-ended legal discovery the NHL seeks will have on the future of my research.

Conclusion

30. The work of the CTE Center and of my scientific collaborators around the

world has produced significant new information about the effects of repetitive concussive and subconcussive impact on an individual's long term health. As discussed in the preceding paragraphs, two separate NINDS sponsored consensus panels of expert neuropathologists concluded that CTE is a distinct disease with defined pathological criteria for diagnosis. They reached this conclusion after reviewing pathology slides from 40 cases of CTE from the BU CTE center. The existence of CTE is not a question for debate. The consensus findings, and those in the published literature, have been of tremendous importance to not only professional athletes, but also to child athletes and members of the military and to those tasked with their care. There remains, however, much research and discovery to be made to further advance the CTE discussion. I fear the impact of an intrusive and over-reaching subpoena, possibly aimed at undermining this entire field of discovery, on the integrity of these future research efforts on this critical topic and, in turn, on any area of research that might impact well-resourced and well-organized litigants. I, along with members of my laboratory, respectfully ask this Court to prevent this very real risk from being realized.

Further affiant sayeth not.

Subscribed and sworn under the penalties of perjury.

Ann McKee, M.D.

Date: February 3, 2017

CURRICULUM VITAE
Ann C. McKee, M.D.

EMAIL AND WEBSITES: amckee@bu.edu, ann.mckee@va.gov, and http://www.bu.edu/cste, www.bu.edu/alzresearch

ACADEMIC TRAINING:

1975 B.S.	University of Wisconsin, Madison, Wisconsin
1979 M.D.	Case Western Reserve School of Medicine, Cleveland, Ohio

POSTDOCTORAL TRAINING:

1979 -1980	Intern in Internal Medicine, Cleveland Metropolitan General Hospital
1980 - 1981	Junior Assistant Resident in Internal Medicine, Cleveland Metropolitan General Hospital
1981 -1982	Senior Assistant Resident in Internal Medicine and Junior Assistant Resident in Neurology, Cleveland Metropolitan General Hospital
1982 -1983	Senior Assistant Resident in Neurology, Cleveland Metropolitan General Hospital
1983 - 1984	Chief Resident in Neurology and Neuropathology, Cleveland Metropolitan General
1984 - 1985	Fellow in the Neurology of Aging, University of Massachusetts Medical Center
1986 - 1988	Chief Resident in Neuropathology, Massachusetts General Hospital
1988 - 1989	Resident in Pathology, Massachusetts General Hospital

ACADEMIC APPOINTMENTS:

1984 - 1985	Clinical Fellow in Neurology, University of Massachusetts Medical Center
1986 - 1989	Clinical Fellow in Neuropathology (Pathology), Harvard Medical School
1989 - 1991	Instructor in Neuropathology (Pathology), Harvard Medical School
1991 - 1994	Assistant Professor in Neuropathology (Pathology), Harvard Medical School
1994 - 2011	Associate Professor in Neurology & Pathology, Boston University School of Medicine
2011 -	Professor in Neurology & Pathology, Boston University School of Medicine

HOSPITAL APPOINTMENTS:

1991 - 1994	Assistant Pathologist (Neuropathology), Massachusetts General Hospital
1994 - 2006	Staff member, Boston Medical Center
1994 -	Staff member, Bedford Veterans Administration Medical Center
2009 -	Staff member, Boston Veterans Administration Medical Center
2010-	Clinical privileges: Boston, Bedford, Togus, White River, Providence, and Manchester VAMCs

AWARDS, HONORS:

1984	Cleveland Neuroscience Research Award
1984	Cleveland Metropolitan General Hospital Resident Research Award
1989	Career Investigator Development Award, sponsored by the National Institute of Neurological and Communicative Disorders and Stroke, KO8 NS 01368
1990	Moore Award, American Association of Neuropathologists
1994	Merit Award, Department of Veterans Affairs, U.S. Government
2006	Moore Award, Honorable Mention, American Association of Neuropathologists
2009	Moore Award, Honorable Mention, American Association of Neuropathologists
2010	Blythe Memorial Lectureship, University of North Carolina Medical Center, Chapel Hill, North Carolina
2010	Massachusetts Neuropsychology Society Keynote Speaker
2010	MacFarlane Conference on Brain Injury Keynote Speaker, Little Rock, Arkansas
2011	Keynote speaker for Alzheimer's Day, Sponsored by the Department of Biochemistry, Boston University School of Medicine, Boston MA
2011	Bostonian of the Year, Boston Globe
2011	Harold Alfond Sports Medicine Lectureship
2011	Gabriele Zu Rhein Lectureship in Neuropathology, University of Wisconsin, Madison
2011	Sports Legacy Institute, Impact Award
2012	Patriot Award, Appleton East High School, given to an alumnus for contributions to the community
2012	Merit Award, Department of Veterans Affairs, U.S. Government
2013	John Groves Lectureship in Neuropathology, McMaster University, Hamilton, Ontario
2013	Zimmerman Lectureship in Neuropathology, Montefiore and Albert Einstein Medical Centers, New York, NY
2013	See the Line, Traumatic Brain Injury Symposium Keynote Speaker, London, Ontario, CA
2013	Davis Lecture at Samford University, Birmingham AL
2013	Adams Lecture, Massachusetts General Hospital, Boston MA
2014	Pamela Warden Lectureship 2104, Defense and Veterans Brain Injury Center, Bethesda, Md
2014	Keynote lecture, Women in Science (WIS) luncheon, Museum of Science, Boston, MA
2014	Ethos Award, Santa Clara University, Institute of Sports Law and Ethics
2014	Keynote Lecture, Ochsner Neuroscience Institute, New Orleans, LA
2014	Deans Distinguished Lecture, 2014, Case Western Reserve University, Cleveland Ohio
2014	Keynote Lecturer, Third Annual Joining Forces TBI/PTSD Boston University School of Medicine and VA Boston Conference
2014	Topical Lecture, 2015, AAAS annual meeting, San Jose California

LICENSES AND CERTIFICATION:

1986 Massachusetts Medical License
1986 American Board of Psychiatry and Neurology
1989 American Board of Pathology - Special Qualification in Neuropathology

TEACHING EXPERIENCE AND RESPONSIBILITIES:

1983 - 1984 Lab instructor in Neuropathology, Case Western Reserve School of Medicine

1984 - 1985 Tutorial course in Clinical Neurology, University of Massachusetts Medical School

1986-1994 Teaching of Neuropathology to Pathology, Neurology, Neurosurgery and Neuroradiology Residents, Massachusetts General Hospital

1986-1987 Clinical instructor in Harvard Medical School course 709.0, Nervous System Pathophysiology 1987-1992
Instructor in Information Processing and Behavior,
Oliver Wendell Holmes Society (New Pathway Program), Harvard Medical School

1994 - present Teaching of Neuropathology to: Medical Students, Pathology Residents, Neurology Residents, Microbiology Students, Dental Students, Graduate Students at the following facilities: Boston University School of Medicine, Mallory Institute of Pathology, Boston Medical Center, Bedford Veterans Administration Medical Center, West Roxbury Veterans Administration Medical Center

1999 - 2006 Lecturer in the Pathological Basis of Disease Course (Boston University Medical students Year 2) – 7 lectures per year
Lecturer in Pathological Basis of Disease Course (Boston University Dental Students) -1 lecture per year
Lecturer in Microbiology (Boston University Medical Students, Year 2) – 2 lectures/year

1994 -present Clinicopathological case presentations: Medical grand rounds – Boston University School of Medicine, Medical and Geriatric Research Educational Clinical Center Neurology Rounds – Bedford Veterans Administration Medical Center, Boston University Alzheimer's Disease Center Weekly Conference, Brain cutting weekly sessions, Bedford and West Roxbury Veterans Administration Medical Centers

MAJOR ADMINISTRATIVE RESPONSIBILITIES:

1996- Director of the Brain Bank, Boston University Alzheimer's Disease Center

1996-	Neuropathology Core Director, Boston University Alzheimer's Disease Center
1997-	Director of Brain Bank, Framingham Heart Study
2001-	Director of Brain Bank, Centenarian Study
2008-	Director of Neuropathology, New England VISN-1 Veteran Affairs Medical Centers
2008-	Neuropathologist, National VA ALS Biorepository
2008-	Co-Director, Center for the Study of Traumatic Encephalopathy, Boston University School of Medicine
2014-	Director of CTE Center, Boston University Alzheimer's Disease Center
2014-	Associate Director, Boston University Alzheimer's Disease Center

OTHER PROFESSIONAL ACTIVITIES:

PROFESSIONAL SOCIETIES: MEMBERSHIPS, OFFICES, AND COMMITTEE ASSIGNMENTS

1987-	American Association of Neuropathologists
2008-	Medical Advisory Board for Sports Legacy Institute (SLI)
2009-	Mackey White TBI Committee for the NFLPA (National Football League Players' Association)
2011	Dana Alliance for Brain Initiatives, Dana Foundation
2012	Co-organizer with Sam Gandy and Steven T DeKosky: Keystone Symposium, Clinical and Molecular Biology of Traumatic Brain Injury and Post-Traumatic Stress Disorder, Keystone Colorado, February 26-March 2, 2012
2012	4th International Consensus Conference on Concussion in Sport, Zurich, Switzerland October 31 -November 2, 2012
2013	National Academies' (National Research Council's) Panel on Human Research and Engineering at the Army Research Laboratory
2013	New York Academy of Science
2014	Co-organizer with Lee Goldstein, Ramon Arrastia-Diaz, Keystone Symposium on Traumatic Brain Injury, Santa Fe, New Mexico, January 2016
2014	Organizer, Consensus Conference to determine the Neuropathological Criteria for CTE, Boston, MA February 2015, Sponsored by NINDS and NIBIB

EDITORIAL BOARDS:

Ad hoc reviewer, Journal *of Neuropathology and Experimental Neurology*
Ad hoc reviewer, *Neurology*
Ad hoc reviewer, *Annals of Neurology*
Ad hoc reviewer, *Neurobiology of Aging*
Ad hoc reviewer, *Arch Neurology*
Ad hoc reviewer, *Brain Research*
Ad hoc reviewer, *Acta Neuropathologica*
Ad hoc reviewer, *Amer J Pathology*

MAJOR COMMITTEE ASSIGNMENTS:

FEDERAL GOVERNMENT

Ad Hoc Reviewer, National Institute of Aging

Neuropathologist and Neurologist, Workshops for the Diagnosis of Progressive Supranuclear Palsy, NINDS, Neuroepidemiology Branch, 1993, 1994, 1996

Vascular Cognitive Impairment Harmonization Criteria Workshop I – Sponsored by the NINDS, Canadian Stroke Network, Canadian Institute of Health Research, Alzheimer's Association, and NIH Office of Rare Diseases, April 24–27, 2005.

Vascular Cognitive Impairment Harmonization Criteria Workshop II– Cell Biology of Vascular Cognitive Impairment Workshop - – Sponsored by the NINDS, Canadian Stroke Network, Canadian Institute of Health Research, Alzheimer's Association, and NIH Office of Rare Diseases, 2007

Traumatic Brain Injury Workshop, Sponsored by NIA and NIH, Bethesda MD, September 18-20, 2009

Sensory and Motor Dysfunction in Aging and Alzheimer's Disease Workshop, Sponsored by NIA and NIH, Bethesda MD, August 8 and 9, 2010

Neuropathology of Chronic Traumatic Encephalopathy Workshop, Sponsored by NIH and NINDS, December 5-6, 2012 Bethesda, Maryland

Organizer, Consensus Conference to determine the Neuropathological Criteria for CTE, Boston , MA February 2015, Sponsored by NINDS and NIBIB

SENATE and CONGRESSIONAL TESTIMONY or BRIEFINGS

October 28, 2009 **Congressional Hearing on the Legal Issues Relating to Football Head Injuries.**http://www.cspan.org/Watch/Media/2009/10/28/HP/A/24819/House+Judiciary+Cmte+Hearing+on+NFL+Head+Injuries.asp

October 6, 2011	**Congressional Briefing on TBI.** Hosted by the Dana Foundation and the AAAS, Rayburn Office building
October 19, 2011	**Hearing before the Senate Committee on Commerce, Science & Transportation: Detection and Treatment of Concussions in Student Athletes** http:// www.**c-span**video.org/program/302174-1
July 20, 2012	**Congressional Briefing: Emerging Research in Head Injuries: What's Happening to our War and Sports Heroes?** Hosted by: The Congressional Men's Health Caucus and the Men's Health Network

CURRENT SUPPORT:

1. *Title:* **Chronic Effects Neurotrauma Consortium (CENC)**
Role in Project: Ann McKee, Co-Director, Neuropathology Core
Type of Grant: Cooperative agreement
Funding Agency: VA, DOD
Years Funded: 1/01/2014-12/30/2019
Total Direct Costs: $500,000/ yr

2. *Title:* **CTE and Posttraumatic Neurodegeneration: Neuropathology and Ex Vivo Imaging**
Role in Project: Ann McKee, PI
Type of Grant: UO1, NIH Cooperative agreement, 1U01NS086659-01
Funding Agencies: National Institute of Neurological Disorders and Stroke (NINDS) National Institute of Biomedical Imaging and Bioengineering (NIBIB)
Years Funded: 12/01/2013-11/30/2017
Total Direct Costs: $ 6,000,000
(Response to NIH RFA: Collaborative Research on Chronic Traumatic Encephalopathy and Delayed Effects of Traumatic Brain Injury: Neuropathology and Neuroimaging Correlation (U01), Cooperative agreement)

3. *Title:* **National VA PTSD Brain Bank**
Role in Project: Ann McKee, Co-investigator
Type of Grant: Congressional mandate
Funding Agency: Veterans Administration, U.S. Government
Total Direct Costs: $500,000/ yr

4. *Title:* **Neuropathology Core, Boston University Alzheimer's Disease Core Center**
Role in Project: Director of Neuropathology Core; Co-Investigator
Type of Grant: P30 Center Grant (N. Kowall, PI) P30-AG13846
Funding Agency: National Institute on Aging
Years Funded: 7/1/1996-6/30/2011
Total Direct Costs: $965,065
The major goal of this project is to build a broad-based research program in Alzheimer's disease to better understand the phenomenology and pathophysiology of Alzheimer's disease and related dementias, and to facilitate treatment and prevention.

5. *Title:* **National VA Biorepository**
Role in Project: Co-investigator, Boston, MA

Type of Grant: VA National Center Grant (N. Kowall, PI)
Funding Agency: Department of Veterans Affairs
Years Funded: 8/01/2009- 7/30/2013
Total Direct Costs: $1,226,000
The major goal of this project is to build a national brain bank to better understand the phenomenology and pathophysiology of ALS.

6. *Title:* **Axonal Injury and Tau Pathology following Blast mTBI in OEF/OIF Veterans**
Role in Project: Ann McKee, PI
Type of Grant: VA Merit Award
Funding Agency: Veterans Administration, U.S. Government
Years Funded: 7/01/2013-6/30/2018
Total Direct Costs: $1,092,348
Program: Tau protein and biomarkers in blast TBI

7. *Title:* **Molecular & Genetic Investigation of Tau in Chronic Traumatic Encephalopathy**
Role in Project: Ann McKee, Co-investigator, Boston, MA
Type of Grant: ERMS#
Funding Agency: Department of Defense
Years Funded: 9/1/14-8/31/17
Total Direct Costs: $119,335
The objective of this grant is to validate the association of CTE with MAPT and characterize differences the expression of tau protein, tau-associated proteins, tau mRNA and tau-silencing microRNAs in CTE patients.

PHILANTHROPIC SUPPORT

Andlinger Foundation 2012: $160,000 to Co-Directors of CSTE (4)

WWE 2013-2015: $1,000,000 over 3 years to McKee, Nowinski and Goldstein

PAST SUPPORT

1. *Title:* **Unrestricted gift- Center for the Study of Traumatic Encephalopathy**
Role in Project: Co-Investigator
Type of Grant: Private Agency
Funding Agency: National Football League
Years Funded: 5/01/2010 – 8/31/11
Total Gift: $1,000,000

2. *Title:* **MRI, Genetic and Cognitive Precursors of AD and Dementia**
Role in Project: Co-Investigator
Type of Grant: RO1 (Phillip Wolf, PI) 2 R01 AG1649
Funding Agency: National Institute on Aging
Years Funded: 6/30/10-05/31/15
Total Direct Costs: $825,557

The goals of the study are to relate risk factors for Alzheimer's Disease (AD) and dementia in the Framingham Offspring and Omni cohorts to changes in brain morphology measured by Magnetic Resonance Imaging (MR) and cognitive performance.

3. *Title:* **Neuropathological and Clinical Consequences of Repetitive Concussion in Athletes**
Role in Project: Co-PI (Robert Stern, Co-PI)
Type of Grant: Private Foundation
Funding Agency: National Operating Committee on Standards for Athletic Equipment (NOCSAE)
Years funded: 2/1/2009-5/1/2011
Total Direct Costs: $249,997
To establish a living brain donation registry, conduct neuropathological examinations, and collect pilot clinical research data, investigating the long-term consequences of repetitive head trauma in athletes.

4. *Title:* **Neuropathologic Examination of Traumatic Encephalopathy in Athletes with Histories of Repetitive Concussion**
Role in Project: Co-PI (Robert Stern Co-PI)
Type of Grant: Supplement to Boston University Alzheimer's Disease Center
Funding Agency: NIA
Years Funded: 7/1/2008-6/30/2009
Total Direct Costs: $100,000
To establish a brain bank, brain donation program, and clinical research program, investigating the long-term consequences of repetitive concussions.

5. Title: **Development of Minimal Diagnostic Criteria for Chronic Traumatic Encephalopathy**
Role in Project: Co-PI (Robert Stern Co-PI)
Type of Grant: Supplement to Boston University Alzheimer's Disease Center
Funding Agency: NIA
Years Funded: 7/1/2009-6/30/2010
Total Direct Costs: $100,000
To develop minimal diagnostic neuropathological criteria for Chronic Traumatic Encephalopathy

6. *Title:* **Establishment of the Center for the Study of Traumatic Encephalopathy**
Role in Project: Co-PI (Robert Stern Co-PI)
Type of Grant: BU Internal Funding
Funding Agency:
Years Funded: 7/1/2008-6/30/2009
Total Direct Costs: $55,000
A formal collaboration with the non-profit Sports Legacy Institute (SLI) establishing the Center for the Study of Traumatic Encephalopathy (CSTE). The Center, jointly located at Boston University School of Medicine (BUSM) and the Edith Nourse Rogers Memorial Veterans Hospital, for studying Chronic Traumatic Encephalopathy (CTE).

7. *Title:* **Role of Somatic mtDNA Mutations in Late-Onset Neurodegeneration**
Role in Project: Co-Investigator

Type of Grant: RO1, Konstantin Khrapko, PI R01 ES11343
Funding Agency: NINDS
Years Funded: 6/1/01 - 5/31/06

8. *Title:* **Tau Hyperphosphorylation in Alzheimer's Disease**
Role in Project: Ann McKee, PI
Type of Grant: Merit Award
Funding Agency: Veterans Administration, U.S. Government
Years Funded: 2/1/94 - 1/31/99
Total Direct Costs: $650,000

9. *Title:* **Microtubule Reorganization in Alzheimer's Disease**
Role in Project: Ann McKee, PI, KO8 NS 01368
Type of Grant: Career Investigator Development Award
Funding Agency: National Institute of Neurological and Communicative Disorders and Stroke
Years Funded: 7/01/89- 6/30/94
Total Direct Costs: $500,000

INVITED LECTURES AND PRESENTATIONS

1996 - present	*Cognitive and Behavioral Neurology: Focus on Dementia, Harvard Medical School CME course,* "Neuropathology of Dementia," Annual Course, Boston MA.
April 24, 2009	*Chronic Traumatic Encephalopathy*, Concussion and Athlete Conference, Franklin Pierce University, Rindge, NH
May 1, 2009	*Chronic Traumatic Encephalopathy*, Sixth Annual Sports-Related Conference on Concussion and Spine Injury, *Harvard Medical School CME course,* Fenway Park, Boston MA
May 19th, 2009	*Chronic Traumatic Encephalopathy in Athletes*, National Football League, Mild Traumatic Brain Injury Committee, New York, NY
September 18, 2009	*Chronic Traumatic Encephalopathy: Progressive tauopathy following repetitive head injury.* Traumatic Brain Injury Workshop, Sponsored by NIA and NIH, Bethesda MD
September 24, 2009	*Recent findings in professional and non-professional football players,* Harvard Faculty Club Breakfast Meeting, Harvard Club, Boston MA
September 29, 2009	*Chronic Traumatic Encephalopathy in Athletes*, Physicians of the Middlesex Central District of the Massachusetts Medical Society, Emerson Hospital, Concord MA
October 2, 2009	*Chronic Traumatic Encephalopathy in Athletes*, Boston University School of Medicine CME course, Concussion and the Athlete conference, Gillette Stadium, Foxborough, MA

October 16, 2009	*Traumatic Encephalopathy*, American Association of Professional Ringside Physicians, Mohegan Sun, Uncasville, CT
October 28, 2009	Congressional Hearing on the Legal Issues Relating to Football Head Injuries.http://www.cspan.org/Watch/Media/2009/10/28/HP/A/24819/House+Judiciary+Cmte+Hearing+on+NFL+Head+Injuries.asp
November 10, 2009	*Chronic Traumatic Encephalopathy Update*, National Football League, Mild Traumatic Brain Injury Committee, New York, NY
January 10, 2010	*Chronic Traumatic Encephalopathy*, Eastern Athletic Trainer's Association, Copley Marriot, Boston MA
January 26, 2010	*Center for the Study of Chronic Traumatic Encephalopathy,* National Football League Players' Association TBI committee, The Breakers, Palm Beach, FL
February 25, 2010	*Chronic Traumatic Encephalopathy, an update*. Roxbury Society for Medical Improvement, The Country Club, Brookline, MA.
March 6-10, 2010	*Chronic Traumatic Encephalopathy in Athletes: the role of axonal pathology,* 2010 Marian Kies colloquium, Indicators of axonal pathology in the CNS, American Society of Neurochemistry, Santa Fe, NM
March 30, 2010	*Chronic Traumatic Encephalopathy. Keynote Speaker,* Blythe Memorial Lecture, University of North Carolina Medical Center, Chapel Hill, North Carolina
April 12, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes.* University of Massachusetts, Lowell Campus, Research Seminar, Lowell, MA
April 15 2010	*What is so Special about our Brain?* Community Action Council, Community Center, Roxbury MA
April 22, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes.* Keynote speaker in Traumatic Brain Injury: Implications for Sport and Health University of Baltimore School of Law and its Center for Sport and the Law, Baltimore MD
April 27, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes and soldiers.* VA Research Day, West Roxbury VAMC, West Roxbury, MA
May 14, 2010	*Chronic Traumatic Encephalopathy*, Seventh Annual Sports-Related Conference on Concussion and Spine Injury, *Harvard Medical School CME course,* Fenway Park, Boston MA

June 2, 2010	*Chronic Traumatic Encephalopathy: a distinct pathological entity associated with head injuries*. NFL- Johns Hopkins Conference on Head Injuries in Football, Baltimore MD
June 8, 2010	*Chronic Traumatic Encephalopathy: a distinct pathological entity associated with head injuries*. Massachusetts Neuropsychology Society, Keynote Speaker, Brookline MA
July 21, 2010	*Chronic Traumatic Encephalopathy.* Keynote Speaker MacFarlane Conference on Brain Injury, Little Rock, Arkansas
August 1, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes* New Jersey Athletic Trainers Association, Princeton, NJ
August 8, 2010	*The Neuropathology of Preclinical Alzheimer's Disease: The Role of the Visual Association Cortex and the Lens*. Sensory and Motor Dysfunction in Aging and Alzheimer's Disease Workshop, Sponsored by NIA and NIH, Bethesda MD
August 26, 2010	*The Long Term Sequelae of Traumatic Brain Injury - What we've learned from athletes: Chronic Traumatic Encephalopathy.* Private presentation to Secretary Shinseki, Bedford VAMC, Bedford MA
August 30, 2010	*The Long-term Sequelae of Mild Traumatic Brain Injury.* DBVIC. Military TBI Training Conference, Washington DC
August 31, 2010	*The Long Term Sequelae of Traumatic Brain Injury - What we've learned from athletes: Chronic Traumatic Encephalopathy.* Private presentation to General Chiarelli, Vice Chief of Staff U.S Army, Pentagon, Washington, D.C.
September 10, 2010	*Major Consequences of Invisible Brain Injury* University of California, San Francisco Neurology Grand Rounds, San Francisco, CA
September 21, 2010	*The Invisible Injury: Mild TBI, Major Consequences.* Annual Keynote Keynote speaker for Alzheimer's Day, Sponsored by the Department of Biochemistry Boston University School of Medicine, Boston MA
October 1, 2010	*Long-Term Effects of Repetitive Concussive and Subconcussive Brain Trauma: Chronic Traumatic Encephalopathy (CTE)* Head Trauma and the Athlete Conference, Waltham, MA
October 1, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Wisconsin Psychiatric Association Meeting, Green Bay WI
October 19, 2010	*The Science of Repetitive Head Trauma (Sub-Concussive Events): Chronic Traumatic Encephalopathy (CTE)* Ice Hockey Concussion Summit. Mayo Clinic Rochester, MN

October 29, 2010	*The Long Term Sequelae of Traumatic Brain Injury - What we've learned from athletes: Chronic Traumatic Encephalopathy.* Private presentation to Rear Admiral Christine Hunter Deputy Director TRICARE Management Activity, Boston University School of Medicine, Boston MA
November 3, 2010	*Decoding the True Impact of Repetitive Brain Trauma/ Chronic Traumatic Encephalopathy/ Encephalomyelopathy* Boston University Alumni Association Breakfast Meeting with President Robert Brown, Standard Club, Chicago IL
November 15, 2010	*The Neuropathological Substrate of MCI*. Boston University Alzheimer's Disease Symposium on Mild Cognitive Impairment, Hebert Lounge, BUSM, Boston, MA
November 16, 2010	*Our Brain in Aging and Disease,* Community Action Council, 12th Baptist Church, Roxbury MA
December 3, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Marshfield Clinic Grand Rounds, Marshfield, WI
December 16, 2010	*Mild TBI, Major Consequences: Chronic Traumatic Encephalopathy/ Encephalomyelopathy*. Walter Reed Army Hospital. Bethesda MD
December 30, 2010	*Chronic Traumatic Encephalopathy: What we have learned from athletes.* Boston Museum of Science featured talk. Boston, MA
February 3, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* International Neuropsychological Society Meeting, Boston, MA
February 4, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Sports-Related Brain Injury Panel Lecture, Nebraska Wesleyan University, Lincoln, NE
February 5, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Mild Traumatic Brain Injury: Challenges and Controversies in Research Conference, Toronto Rehabilitation Center, Toronto ON
February 15, 2011	*Major Consequences of Invisible Brain Injury: Chronic traumatic encephalopathy and encephalomyelopathy.* University of Massachusetts Grand Rounds, Worcester, MA
March 3, 2011	*Chronic Traumatic Encephalopathy: What we have learned* Harold Alfond Sports Medicine Lecture, University of New England Biddeford Maine
March 16, 2011	*Chronic Traumatic Encephalopathy* The Regeneration of Brain Synapses: Science, Implications and Opportunities Conference, Bethesda, MD

March 30, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* University of New Mexico Grand Rounds, Albuquerque, NM
April 15, 2011	*The Pathology of Chronic Traumatic Encephalopathy* Cumulative Sports Concussion and Risk of Dementia American Academy of Neurology Annual Meeting, Honolulu, HI
April 17, 2011	I. Acute *Concussive and Subconcussive injury* II. *Chronic Traumatic Encephalopathy: What we have learned from athletes* Traumatic Brain Disorders - Neurobehavioral Assessment and Management Conference, Columbus, OH
April 29, 2011	University of Edinboro Concussion Conference, Edinboro, PA
May 11, 2011	*Chronic Traumatic Encephalopathy: Your brain on football.* Keynote speaker, Christ Medical Center Neurosciences Institute, Oak Lawn, IL
June 19, 2011	*Chronic Traumatic Encephalopathy* National Athletic trainers Association Annual Meeting, New Orleans, LA
June 23, 2011	Suna Kıraç Conference on Neurodegeneration, ALS-IST 2011: *Recent Themes in Motor Neuron Biology and Neurodegeneration*, Istanbul, Turkey
July 12, 2011	*Chronic Traumatic Encephalopathy* International Symposium on Brain Injury in Children Conference, Hospital for Sick Children, Toronto, ON
August 16, 2011	*Traumatic Brain Injury* Neurobiology of Brain Dysfunction, Marine Biological Laboratory, Woods Hole, MA
August 22, 2011	*The Long-term Sequelae of Mild Traumatic Brain Injury.* DVBIC. Military TBI Training Conference, Washington DC
September 8, 2011	NIH ALS Conference: Clinical Research to Find the Pathogenesis and Cause of ALS, Tarrytown New York
September 21, 2011	*Chronic Traumatic Encephalopathy* Institute for Memory Impairments and Neurological Disorders Frontiers of the MIND Keynote Speaker, University of California Irvine, CA
September 24, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Neurotrauma Research Day, University of British Columbia
October 6, 2011	Capitol Hill briefing on TBI, sponsored by the Dana Foundation and the AAAS, Rayburn Office building
October 7, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Stroke Rehab Center Conference, Inova Mount Vernon Hospital, Alexandria, VA

October 21, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Neurology Grand Rounds, Temple University, Philadelphia, PA
October 21, 2011	*Recurrent Athletic mTBI, Cerebral Tauopathy, and Chronic Traumatic Encephalopathy* Seventh Annual Lachman Series, Pennsylvania Orthopedic Society, Philadelphia, PA
October 28, 2011	*Long-Term Effects of Repetitive Concussive and Subconcussive Brain Trauma: Chronic Traumatic Encephalopathy (CTE)* Head Trauma and the Athlete Conference, Framingham, MA
October 29, 2011	*Mild Traumatic Brain Injury and Chronic Traumatic Encephalopathy: What we have learned from athletes* Annual Education Conference, Brain Injury Association of Illinois, Oakbrook, IL
November 2, 2011	Sports Injury and Alzheimer's disease: Is there a Connection? Rhode Island Alzheimer's Association Lecture, Providence, RI
November 3, 2011	Gabriele Zu Rhein Lectureship in Neuropathology, University of Wisconsin at Madison
November 16, 2011	*Chronic Traumatic Encephalopathy* President's Lecture Series on Health, Regis College, Wellesley, MA
November 20, 2011	*Chronic Traumatic Encephalopathy: What we have learned from athletes* University of Toronto Department of Laboratory Medicine and Pathobiology Neuropathology Day, Toronto, ON
December 1, 2011	*The Long-term Consequences of Repetitive Concussion*, Boston University Academy, Boston MA
January 7, 2012	*Chronic Traumatic Encephalopathy* Eastern Athletic trainers Association Annual Meeting, New Orleans, LA
February 16, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Neurology Grand Rounds, University of Pennsylvania, Philadelphia, PA
February 22, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Neuroscience and Behavior Program, University of Massachusetts, Amherst, MA
February 27, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes* Keystone Symposium, Clinical and Molecular Biology of Traumatic Brain Injury and Post-Traumatic Stress Disorder, Keystone, CO
March 5, 2012	*The Long-term Consequences of Repetitive Concussion*, Appleton High School East, Appleton, Wisconsin
April 2, 2012	*Chronic Traumatic Encephalopathy,* Wisconsin coroners and medical examiners, Appleton Wisconsin

April 17, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes and Veterans,* Columbia-Presbyterian Neurology Grand Rounds, New York, New York
May 18, 2012	*Chronic Traumatic Encephalopathy* Sports-related injury on Concussion and Spine Injury conference, Children's and Brigham and Women's Hospitals, Boston, MA
May 25, 2012	*Chronic Traumatic Encephalopathy,* Loyola School of Medicine, Chicago Illinois
June 21, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes and Veterans* Plenary speaker, American Association of Neuropathologists annual meeting 2012, Chicago IL
July 25, 2012	*Chronic Traumatic Encephalopathy* Plenary speaker, Neurotrauma Society annual meeting 2012, Phoenix, AZ
August 5, 2012	*Chronic Traumatic Encephalopathy 2012*, American Psychological Association, Orlando FL
August 15, 2012	*Chronic Traumatic Encephalopathy in the Military* Plenary speaker, Military Health System Research Symposium annual meeting 2012, Fort Lauderdale, FL
August 16, 2012	*Chronic Traumatic Encephalopathy: What we have learned from athletes and Veterans* Breakout session speaker, Military Health System Research Symposium annual meeting 2012, Fort Lauderdale, FL
September 28, 2012	*Chronic Traumatic Encephalopathy 2012* Mayo Clinic Symposium on Concussion in Sport, Scottsdale, AZ
October 1, 2012	*Neuropathological Spectrum of Chronic Traumatic Encephalopathy* Lou Ruvo Symposium on Chronic Traumatic Encephalopathy, Cleveland Clinic, Las Vegas NV
October 5, 2012	*Chronic Traumatic Encephalopathy 2012* American Neurological Association annual meeting, Lunch Discussion, Boston MA
October 6, 2012	*Chronic Traumatic Encephalopathy 2012* American Neurological Association annual meeting, Breakout Session on Behavioral Neurology, Boston MA
October 23, 2012	*Chronic Traumatic Encephalopathy 2012* AAAS-DANA Foundation Event on "The Science and Impact of TBI", Washington, DC
October 25, 2012	*Chronic Traumatic Encephalopathy 2012* Hit Count Conference, Waltham MA

October 26, 2012	*Chronic Traumatic Encephalopathy 2012* Boston University Concussion in Sport Conference, Waltham MA
November 2, 2012	*What is the pathological evidence for concussion related changes in the brains of retired athletes?* 4th International Consensus Conference on Concussion in Sport, Zurich, Switzerland November 2, 2012
November 23, 2012	*Chronic Traumatic Encephalopathy 2012* Seminar on Boxing and the Brain, The Royal College of Surgeons, Stephens Green, Dublin Ireland
December 5, 2012	*Chronic Traumatic Encephalopathy-Insights from the brains of athletes* NIH Workshop on the Neuropathology of CTE, Bethesda MD
December 10, 2012	*Chronic Traumatic Encephalopathy 2012* Neurology Grand Rounds, Washington University St. Louis, MO
February 13, 2013	*Concussions to Chronic Traumatic Encephalopathy: what is the evidence?* Merrimack College, North Andover MA
March 13, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy* Neurosurgery Grand Rounds, Massachusetts General Hospital, Boston MA
April 11, 2013	*Brain Damage and Recovery* Judicial Seminar on Emerging Issues in Neuroscience, Sponsored by the American Association for the Advancement of Science, the Federal Judicial Center, the National Center for State Courts, the American Bar Association Judicial Division, and the Dana Foundation, and hosted by the Yerkes National Primate Research Center of Emory University, Atlanta, Georgia
April 19, 2013	*Chronic post-traumatic encephalopathy in professional sport*, John Groves Lectureship in Neuropathology, 6th Annual Neuropathology Day at McMaster, Hamilton, Ontario, Canada
May 5, 2013	*Ocular Manifestations of Chronic Traumatic Encephalopathy*, Association for Research in Vision and Ophthalmology annual meeting, Seattle, WA
May 9, 2013	*Chronic Traumatic Encephalopathy 2013*, Harry Zimmerman Lectureship in Neuropathology, Montefiore Albert Einstein Medical Centers, New York, New York
May 17, 2013	*Chronic Traumatic Encephalopathy*, Advanced Neuro Intensive Care, Harvard Medical School CME course, Boston MA
May 18, 2013	*Update on Chronic Traumatic Encephalopathy,* Sports-related injury on Concussion and Spine Injury conference, Children's and Brigham and Women's Hospitals, Boston, MA

May 18, 2013	*Emerging Concepts in Research on Chronic Traumatic Encephalopathy* VA Research Day, Boston, MA
May 23, 2013	*Chronic Traumatic Encephalopathy in Athletes and Veterans,* American Association of Clinical Scientists, Boston, MA
May 29, 2013	*Interneuronal spreading of tau pathology in chronic traumatic encephalopathy,* PRION 2013: Conquering Frontiers, Banff, Alberta, Canada
July 9, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, 3rd Biennial Conference – Brain Injury in Children, Toronto, Canada
August 14, 2013	*Chronic Traumatic Encephalopathy*, See the Line- Traumatic Brain Injury Symposium, Western School of Medicine, London, Ontario
September 20, 2013	*Current Challenges in Chronic Traumatic Encephalopathy and Mild Traumatic Brain Injury*. Accelerating Translational Neurotechnology: Fourth Annual Aspen Brain Forum Aspen, CO
October 8, 2013	*Prospective Study of CTE using Neuro Imaging with Post Mortem as a Final Validation/Challenges.* Ice Hockey Summit II. Mayo Clinic, Rochester, Minnesota
October 11, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, Davis Lecture at Samford University, Birmingham AL
November 18, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, Vanderbilt University, Nashville Tennessee
November 20, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, Brigham and Women's Neurology Grand Rounds, Boston MA
November 21, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, Adams Lecture, Massachusetts General Hospital, Boston, MA
December 3, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, University of Tennessee Health Sciences Center, Memphis, Tennessee
December 13, 2013	*Emerging Concepts in Chronic Traumatic Encephalopathy*, Mayo Clinic Jacksonville, Jacksonville, Florida
March 12, 2014	*Update on CTE*, Alzheimer's Disease Center Lecture, Boston University, Boston MA
April 3, 2014	*Consequences of mTBI in athletes and military veterans*, Pamela Warden Lectureship 2104, Defense and Veterans Brain Injury Center, Bethesda,

	MD
April 4, 2014	*Update on CTE in athletes*, Wisconsin Athletic Trainers' Association Annual Meeting and Symposium, Eau Claire WI
April 30, 2014	*Update on CTE research*, SLI Family Huddle & McHale Memorial, Washington DC
May 3, 2014	Pathology of CTE, American Association of Neurologists Annual Meeting, Philadelphia PA
May 15, 2014	What is CTE? University of Wisconsin- Founders Day, Boston MA
May 20, 2014	Current concepts in CTE, Ochsner Neuroscience Institute, New Orleans, LA
May 23, 2014	*Update on Chronic Traumatic Encephalopathy,* Sports-related injury on Concussion and Spine Injury conference, Children's and Brigham and Women's Hospitals, Boston, MA
April 7, 2014	*What is CTE?* Keynote lecturer for annual Women in Science (WIS) luncheon, Museum of Science, Boston, MA
June 9, 2014	*Opthalmological Manifestations of CTE*, American Association of Neuropathlogists, Portland, OR
September 15, 2014	*CTE: current concepts*, TBI Society of Brazil, Sao Paulo, Brazil
September 17, 2014	*CTE and the military*, XVIII International Congress of Neuropathology, Rio de Janeiro, Brazil
September 18, 2014	*CTE and athletes*, Neurological Grand Rounds, Good Samaritan Hospital, Sao Paulo, Brazil
September 19, 2014	*CTE –emerging concepts,* Neurological Grand Rounds, University of Sao Paulo Medical School, Sao Paulo, Brazil
October 16, 2014	*Emerging Concepts in CTE*, Deans Distinguished Lecture, Case Western Reserve University, Cleveland, OH
November 4, 2014	*Update on Chronic Traumatic Encephalopathy* , Keynote Lecturer, Third Annual Joining Forces TBI/PTSD Boston University School of Medicine and VA Boston Conference, Boston University
November 7, 2014	*Update on Chronic Traumatic Encephalopathy,* Elon University, North Carolina

November 11, 2014 *CTE in athletes*, XXIV Congresso Brasileiro de Neurologica, Curitiba, Brazil

November 14, 2014 *Update on Chronic Traumatic Encephalopathy* University of New England, Providence Rhode Island

November 20, 2014 *CTE: Emerging Concepts* Texas Neurological Society, Houston, Texas

November 21, 2014 *Football and CTE* 4th Annual Huffines Discussion. Texas A&M, Houston Texas

December 9, 2014 *CTE in football players: What we've learned over the past 7 years.* Neurology Grand Rounds, Boston University School of Medicine, Boston MA

December 12, 2014 *Update on Chronic Traumatic Encephalopathy*, University of Vermont, Neurology grand rounds

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4. **McKee AC**, Kowall NW, Kosik KS: Hippocampal neurons predisposed to neurofibrillary tangle formation are enriched in type II Ca/calmodulin-dependent protein kinase. J Neuropathol Exp Neurol, 1990; 49: 49-63.

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13. Hauw JJ, Daniel SE, Dickson D, Horoupian DS, Jellinger K, Lantos PL, **McKee AC**, Tabaton M, Litvan I. Preliminary NINDS Neuropathological Criteria for Steele-Richardson-Olszewski Syndrome (Progressive Supranuclear Palsy). Neurology; 1994, 44(11): 2015-9.

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CASE REPORTS, REVIEWS, CHAPTERS AND EDITORIALS

Editorials and Critical Reviews

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Exhibit B: VA-BU-CLF Brain Bank Protocol

VA-BU-CLF Brain bank

The brain bank receives the fresh brain and spinal cord tissue on wet ice by courier.

Gross photographs

The VA-BU-CLF brain bank uses a strictly standardized protocol for processing brains that includes comprehensive photographic documentation, histological processing and immunostaining of the brain and spinal cord. Neuropathologists inspect the brain, calvarium, meninges and spinal cord grossly. They photograph the external surfaces of the brain and spinal cord. The midbrain is sectioned transversely; the cerebellum sectioned sagittally; and the cerebral hemispheres are sectioned coronally. The hemisphere is cut into 1.0 cm sections and the posterior surfaces of all sections are photographed from all directions resulting in approximately 100 gross photographs per donor.

Fresh hemisphere

The brain is weighed and the spinal cord measured, the CSF and plasma are aliquoted into 1cc aliquots and stored at -80^{o}C in one of 12 freezers. The fresh brain is hemisected sagittally, the cerebral hemisphere is cut into 0.5 cm coronal slabs, each slab is photographed individually (resulting in 100 photographs of the frozen hemibrain) and immediately snap frozen according to the protocols of the VA-BU-CLF brain bank. Small sections (1 X 1 X 0.5cm) are dissected from the fresh tissue slabs from 36 areas of interest of the brain, cerebellum, brainstem and spinal cord, and one eyeball, and immediately frozen in liquid nitrogen, labeled and stored at -80C. The remainder of the coronal tissue slabs and other tissue are snap frozen on aluminum plates and stored at -80^{o}C. The frozen tissue slabs, small regional biospecimens, CSF and plasma are retained at the VA-BU-CLF brain bank for future distribution to approved investigators.

Fixed hemisphere

The other cerebral hemisphere, half brainstem, cerebellar hemisphere, eye, and remainder of spinal cord are fixed in cold periodate-lysine-paraformaldehyde for 2 weeks, then coronally sectioned, each fixed slab is photographed, resulting in another 100 photographs per case, and tissue sections from 33 regions are dissected and placed into individual cassettes to be embedded in paraffin blocks.

Tissue processing and staining

The 33 paraffin blocks are subsequently processed and cut at 10 microns into 20 tissue sections per paraffin tissue block (*660 paraffin-embedded tissue sections per case*) and the 660 tissue sections are placed on a glass slides for histological and immunocytochemical staining (*660 glass slides per case*). Histological immunocytochemical staining on the glass slides takes approximately 2 weeks to produce. Once the immunocytochemical and histological staining is completed on the glass slides, they are manually cover-slipped and labeled, and placed into slide trays for the neuropathologist to read.

Microscopic diagnosis

The neuropathologist uses a microscope to read the glass slides and a camera mounted to the microscope to take multiple photographs of each glass slide, this takes several days and results in at least an additional 200 photomicrographs per case. The neuropathological data is recorded into a database (2 hours), and a neuropathological report is generated (2 hours)**.** The photographs of the brain and spinal cord and microphotographs are labeled and stored at the VA. The glass slides are also stored at the VA.

Preparation of large landscape slides

Fixed hemispheric sections of brain are stored in ice cold 4% paraformaldehyde-lysine-periodate (PLP) (pH 7.4) followed by sucrose/sodium azide cryopreservation and sectioning at 50μ on a large freezing microtome. The cut tissue sections are immunostained individually (a process requiring a week) and individually mounted by hand on large glass slides. This is an extremely labor-intensive process requiring a total of 3 weeks per case.

#M7FWL0680D1CDFv1

We have approximately 40,000 large glass slides from the brain donors.

Paraffin-blocks prepared per case

	Region
1.	Olfactory bulb
2.	Midbrain at level of red nucleus
2A.	Midbrain at superior cerebellar peduncle
3.	Precentral, postcentral cortex (BA 4,3,2,1)
4.	Inferior parietal cortex (BA 39,40)
5.	Anterior cingulate (BA 24)
5A.	Superior frontal (BA 8,9)
6.	Inferior frontal cortex (BA 10,11,12)
7.	Dorosolateral frontal (BA45, 46)
8A.	Caudate, putamen, and accumbens (CAP), septal cortex, fornix
8B.	Insular cortex
9.	Temporal pole (BA 38)
10.	Superior temporal (BA 20, 21,22)
11.	Amygdala, with entorhinal cortex (BA 28)
12.	Globus pallidus, insula, sub. Innominata
13.	Anterior hippocampus
14.	Hippocampal formation, lateral geniculate
15.	Superior temporal posterior (BA 41,42)
16.	Thalamus with subthalamic nucleus
16A	Hypothalamus, mammillary body
16B.	Posterior thalamus
17.	Posterior cingulate (BA23, 31)
18.	Calcarine cortex (BA 17,18)
19.	Superior parietal cortex (BA 7B)
20.	Upper pons (level of locus cœruleus)
20A.	Pons, middle cerebellar peduncle
21.	Medulla oblongata with inferior olives)
22-1.	Cervical spinal cord
22-2,3	Thoracic spinal cord
22-4, 5	Lumbar spinal cord
22-6	Sacral spinal cord
23.	Cerebellar vermis
24.	Cerebellum with dentate nucleus
25.	Parastriate cortex

Key sheet of blocking and staining procedures

	Region	LHE	Biel	AT8	Aß	□s	TDP-43	P62	APP	SMI 34	IBA1
1.	Olfactory bulb	X	X	X		X	X	X			
2.	Midbrain at level of red nucleus	X		X		X	X	X			X
2A.	Midbrain at superior cerebellar peduncle	X		X		X					
3.	Precentral, postcentral cortex (BA 4,3,2,1)	X		X			X	X	X	X	X
4.	Inferior parietal cortex (BA 39,40)	X		X		X	X	X	X	X	X
5.	Anterior cingulate (BA 24)	X		X							
5A.	Superior frontal (BA 8,9)	X		X			X	X	X	X	X
6.	Inferior frontal cortex (BA 10,11,12)	X		X	X		X	X	X	X	
7.	Dorosolateral frontal (BA45, 46)	X		X	X	X	X	X	X	X	X
8A.	Caudate, putamen, and accumbens (CAP), septal cortex	X		X			X	X			X
8B.	Insular cortex	X		X			X	X			X
8C.	Fornix	X		X			X	X	X	X	X
9.	Temporal pole (BA 38)	X		X			X	X			X
10.	Superior temporal (BA 20, 21,22)	X		X	X	X	X	X			X
11.	Amygdala, with entorhinal cortex (BA 28)	X		X		X	X	X			X
12.	Globus pallidus, insula, sub. Innominata	X		X			X	X			
13.	Anterior hippocampus	X	X	X		X	X	X			X
14.	Hippocampal formation, lateral geniculate	X	X	X	X	X	X	X			X
15.	Superior temporal posterior (BA 41,42)	X		X					X	X	
16.	Thalamus with subthalamic nucleus	X		X		X	X	X			
16A	Hypothalamus, mammillary body	X		X			X	X			X
16B.	Posterior thalamus	X		X				X	X	X	X
17.	Posterior cingulate (BA23, 31)	X		X	X						
18.	Calcarine cortex (BA 17,18)	X		X	X						
19.	Superior parietal cortex (BA 7B)	X		X	X			X	X	X	
20.	Upper pons (level of locus cœruleus)	X		X		X	X	X	X	X	X
20A.	Pons, middle cerebellar peduncle	X		X							
21.	Medulla oblongata with inferior olives)	X		X		X	X	X			
22-1.	Cervical spinal cord	X		X				X			X
22-2,3	Thoracic spinal cord	X		X				X			
22-4, 5	Lumbar spinal cord	X		X				X			X
22-6	Sacral spinal cord	X		X				X			
23.	Cerebellar vermis	X		X				X			
24.	Cerebellum with dentate nucleus	X		X			X	X	X	X	X
25.	Parastriate cortex	X		X	X			X			

Yolanda Sherman

From: ecf-notice@mnd.uscourts.gov
Sent: Monday, February 06, 2017 3:59 PM
To: mndecfnotifications@mnd.uscourts.gov
Subject: Activity in Case 0:14-md-02551-SRN-JSM IN RE: National Hockey League Players' Concussion Injury Litigation Affidavit

U.S. District Court

U.S. District of Minnesota

Notice of Electronic Filing

The following transaction was entered by Elswit, Lawrence on 2/6/2017 at 2:58 PM CST and filed on 2/6/2017
Case Name: IN RE: National Hockey League Players' Concussion Injury Litigation
Case Number: 0:14-md-02551-SRN-JSM
Filer: Trustees of Boston University/CTE Center
Document Number: 682

Docket Text:
AFFIDAVIT of Ann McKee re [680] Memorandum in Opposition to Motion, by Trustees of Boston University/CTE Center. (Attachments: # (1) Exhibit(s), # (2) Exhibit(s))(Elswit, Lawrence)

0:14-md-02551-SRN-JSM Notice has been electronically mailed to:

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